Modern Concepts of Cardiovascular Disease

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TRAUMATIC HEART DISEASE*

The effects of trauma upon the heart and great vessels may be considered in the light of the immediate consequences of the trauma itself, or the delayed effects of pathological lesions produced. Among the immediate effects are massive external or intrapleural hemorrhage, cardiac tamponade, and peripheral embolism by penetrating foreign bodies. Among the late manifestations are constrictive pericarditis, ventricular and aortic aneurysms, cardiac decompensation from traumatically produced shunts in the heart or in the great vessels, and complications due to foreign bodies in the heart or pericardium.

Massive hemorrhage from wounds of the heart or great vessels, whether external or into the pleura, results in rapid exsanguination. Penetrating wounds of the aorta or pulmonary artery, due to stab wounds and bullet wounds, have been thought to result in immediate exsanguination, but isolated instances of suture, with survival, have been reported for a good many years. A recent report from Texas cites 23 patients with perforating wounds of the aorta, of whom seven were saved and survived indefinitely. The wound in mediastinal pleura or pericardium may offer enough impediment to the outflow of blood, in some instances, to allow time for lives to be saved by prompt thoracotomy and direct suture.

There have been numerous instances of successful suture of wounds of the atria, and occasionally of the ventricles, with free communication to the pleural cavity or the exterior. However, the operation, through a formal thoracotomy incision, must be performed immediately. In the present era of cardiac surgery, simple suture of the heart does not merit the detailed attention which would have been accorded it in an article such as this 15 years ago. One layer of continuous silk sutures, passing under any coronary artery which happens to be adjacent to the laceration, is all that is required.

From the standpoint of the therapeutic effort, it is essential to distinguish between a patient suffering from a stab or bullet wound of the heart, with collapse from massive hemorrhage, and a patient with similar injury in collapse from pericardial tamponade. The differentiation may be made, apart from the lack of evidence of massive hemothorax or external blood loss, by the appearance in the patient with cardiac tamponade of arterial hypotension, visible engorgement of the veins of the neck, measurable elevation of venous pressure, distant or inaudible heart sounds, absent precordial pulsations, and, under the fluoroscope, inconspicuous cardiac pulsations. The pericardium is not readily distended and an increase in volume within the pericardium requires days to appear, so that the percussable outline of cardiac dullness and the radiological shadow are not increased. Many patients with pericardial tamponade have a striking degree of irrational excitement from hypoxia, of a kind rarely manifested in patients with an equal degree of arterial hypotension purely from blood loss.

The pathophysiology of cardiac tamponade has been well studied by Isaacs, Martin, Schenk, and others. The accumulation of fluid under pressure in the pericardium exerts its most significant effect by interference with the proper filling of the ventricles. Contrary to previous opinion, the obstruction does not exert its influence principally upon the entering veins and the atria, but most significantly upon the ventricles. The physiological effects can be reproduced experimentally by selective tamponade of the ventricles. The result is decreased cardiac output and death. Of great significance is the observation that as cardiac output falls, venous pressure rises before arterial pressure falls, presumably because peripheral vasoconstriction can maintain arterial

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pressure for a time in the face of decreased output. The elevation of the venous pressure represents back pressure, but is, in itself, a compensatory phenomenon, helping to fill the compressed heart. It has been shown that in patients and animals with cardiac tamponade intravenous infusion of blood is of benefit, in spite of an elevated venous pressure. Of fundamental importance in understanding the rationale for the treatment of cardiac tamponade is awareness of the character of the pressure curves in poorly distensible bladders filled under pressure. With the first increment of fluid into the pericardium, there is relatively little rise in intrapericardial pressure. As the limits of distensibility of the pericardium and compressibility of the heart are reached, the intrapericardial pressure rises very sharply with successive introductions of small increments of fluid. By the same token, if at this point small amounts of fluid are withdrawn from the tensely distended pericardium, there is, at first, a sharp drop in pressure. Withdrawal of as little as 15 ml. may bring striking relief of tamponade in some patients, presumably those in whom the myocardial wound has sealed itself.

Careful evaluation of large series of patients with penetrating wounds of the heart has indicated that the patients with cardiac tamponade who reach the hospital emergency room alive are a selected group. Those with massive wounds of the heart have usually succumbed to external hemorrhage or to insupportable tamponade. The patients reaching the hospital alive with tamponade are usually those who have relatively small wounds of the ventricles or atria. The result is an equilibrium between intrapericardial pressure and the flow of blood from the injured heart, perhaps aided by a small, indirect decompressing tract to the outside, or to the mediastinum or pleural cavity, so that fatal compression of the heart does not occur for some time. Such wounds are sufficiently small so that if the patient can be briefly tided over the tamponade the myocardial wounds will clot and heal of themselves. Our own experience would indicate that by the time most patients reach the hospital with pericardial tamponade from perforating wounds, active bleeding from the injured heart has actually ceased. The relative ease with which the myocardium of the ventricle, particularly, can seal a wound was easily demonstrated during the period of treatment of isolated valvular pulmonic stenosis by transventricular valvulotomy. After repeated transventricular instrumentation of the valve with probes, knives, and dilators, a few minutes of simple finger pressure on the myocardial wound will often suffice to stanch the bleeding entirely, although it is usually the custom to insert a precautionary suture or two.

Fifteen to twenty years ago, when antibiotics were fewer and less effective, when anesthesia was substantially less advanced than it is today, and when qualified thoracic surgeons were not so widely available, it was possible to point to a substantial advantage in mortality and morbidity when patients with cardiac tamponade, treated solely by aspiration of the pericardium, were compared with those treated immediately by operation. Today, the difference, particularly in mortality, is probably not as striking as it was formerly. However, it must still be considered undesirable to operate routinely for a condition which often can be treated safely and effectively without general anesthesia and thoracotomy.

Management of the Patient with Pericardial Tamponade

The patient with suspected pericardial tamponade should have arterial and venous pressure constantly monitored and, if possible, be intermittently examined under the fluoroscope. In our emergency department, such patients are placed at once on the fluoroscopic table on which all procedures and studies are performed. Blood transfusion is started at once. If there is no evidence of massive hemothorax or external blood loss and the signs of pericardial tamponade given above are present, or indeed if tamponade seems at all likely, an 18-gauge needle is inserted into the pericardium, either through the fourth interspace to the left of the sternum or by the somewhat safer subxiphoid route. As the needle is inserted, the plunger of the syringe is kept under tension, and the advancement of the needle arrested as soon as blood appears in the syringe. If this is done gently, and no blood is found within the pericardial sac, one will feel the heart pulsate when the pericardial cavity has been traversed. Pericardial blood, defibrinated by the beating of the heart, will usually remain liquid after aspiration. As much blood as possible is aspirated, and the effect upon the arterial pressure, venous pressure, heart sounds, pericardial pulsations, and the patient's state of consciousness and unrest is observed. In most instances, the first aspiration, which may yield anywhere from 15 to 150 ml. of blood, will result in a striking improvement in the arterial pulse and pressure, a more gradual decrease in the venous pressure, an immediate return of audible heart sounds and visible fluoroscopic pulsations, and a change from a cold, clammy, wildly restless patient to one who becomes warm, dry, conscious, and cooperative. If this improvement is maintained, with no further change in the patient's condition, and there is no question of a coincident intra-abdominal injury, the patient is maintained under close observation but is given no further specific treatment. If the symptoms recur, a second aspiration may be attempted and will frequently be all that is required. How-

ever, if symptoms of tamponade return after a second or at most a third aspiration, thoracotomy should be undertaken. To avoid delay, the operating room is notified when the patient is first seen, so that if the decision to operate is made, no time will have been lost.

At the Johns Hopkins Hospital, survivals of patients reaching the hospital alive with recognized cardiac tamponade have been 39 out of 40. Of the 31 patients treated solely by aspiration, none died. The late course of patients successfully treated by pericardial aspiration has given no cause for concern. Suppurative pericarditis has not occurred in our experience, nor have ventricular aneurysms. An occasional instance of constrictive pericarditis has developed (2 cases out of 20, followed from 5 to 20 years), a small price to be paid for the immediate safety and effectiveness of the aspiration treatment of cardiac tamponade.

Traumatic Rupture of the Aorta

A type of acute traumatic disease of the great vessels, of which there has been increasing awareness, is rupture of the thoracic aorta. In association with blunt force from crushing wounds of the chest, with perforating wounds of the aorta and great vessels from the sharp ends of fractured ribs, or in violent deceleration, as in airplane crashes, there may be ruptures of the aorta, most characteristically just distal to the origin of the left subclavian artery. Obviously, rupture of the aorta communicating freely with the pleural cavity results in death, almost instantly. What is not so well appreciated is that with intact mediastinal pleura, a substantial rent in the thoracic aorta, or even a complete transection of the thoracic aorta with separation of the two ends for several centimeters may occur without symptoms as profound as might be expected, so that frequently the condition is not diagnosed. Severe back pain and at least initial shock from which the patient may recover are usually seen. The diagnosis may be suspected from the broadening of the mediastinal shadow, which, when marked, is pathognomonic. Intense pain in the chest or back, unaffected by respiration, should arouse suspicion. While the hematoma in the mediastinum may be very large, it rarely results in occlusion of the distal aorta, and signs of paraplegia or absence of the femoral pulses are not required for diagnosis. Perhaps the most surprising feature of traumatic rupture of the aorta is the apparent frequency with which such lesions pass unrecognized, only to have the patient either succumb to rupture, hours or days later, or appear with an obvious traumatic aneurysm, months or years later.

The immediate treatment, if the lesion is diagnosed, is thoracotomy and repair of the aorta. It is important to dissect the aorta carefully, since what appears to be a small rent may actually be no more than the tear in the adventitia, the other coats being much more extensively injured or even completely transected. The probable necessity for occlusion of the aorta would make it preferable to have facilities available for left atrial-femoral artery bypass. The uncertain period of time required for preparation may determine whether the operation is delayed until arrangements for a bypass are made. The use of hypothermia will decrease the hazard of aortic occlusion in the instances in which bypass is not feasible. It is of interest that in the traumatic aneurysms discovered at a time remote from the injury, rupture has not been known to occur, as opposed to the situation in luetic and arteriosclerotic aneurysms. If this observation proves to be valid, the present therapy of resection of traumatic aneurysms and reconstruction of the aorta may prove to be unnecessary.

Foreign Bodies in the Heart

In traumatic surgery in civilian life, foreign bodies penetrating and remaining in the heart, pericardium, or great vessels, are a relative rarity. Shootings in civilian life are usually at short range and result in through-and-through wounds. The small number of bullet wounds of the chest, compared with the vast number seen during war (most of which were due to irregularly shaped shell fragments) leads to the infrequent occurrence of the combination of circumstances resulting in the presence of a foreign body within the heart or great vessels. Foreign bodies within the cardiac shadow may lie in the pericardium, within the myocardium, or within the chambers of the heart. It may be difficult to make any preoperative distinction, except in the case of a foreign body which is seen to move about and therefore must be loose within the chamber of the heart. Military experience has dealt entirely with the late removal of foreign bodies, symptomatic by virtue of infection, recurring pericardial effusion, or pain. There is some evidence that a foreign body, caught within the trabeculae of the myocardium, may, by constant friction, thin out the overlying heart wall. Embolism in either greater or lesser circulation from foreign bodies, originally loose in the heart or great vessels, may produce severe or fatal complications. The removal of foreign bodies within the chambers of the heart is also indicated on this basis. Very small foreign bodies, which appear to be in the myocardium or pericardium and are not symptomatic, should probably not be disturbed. The presence of a foreign body within the heart shadow does not constitute an indication for emergency intervention; if no other indication exists for immediate operation, time may be

taken for detailed evaluation of the position of the foreign body and to make a decision as to the need for operation.

Arteriovenous Fistula

The type of traumatic heart disease occurring most frequently, indeed going back to antiquity, is heart failure based upon the effects of a long-standing arteriovenous fistula. The physiological effects of a peripheral arteriovenous fistula have long been well known: widened pulse pressure, increased blood volume, and increased cardiac output. Over the lesion itself there is a palpable continuous thrill, and a continuous, raucous machinery murmur. Branham's phenomenon, decrease in the heart rate and in the pulse pressure, results when the communication is occluded. Large and dilated veins may appear in the region of the fistula. Distal nutrition of the part is unaffected. While small fistulae impose little cardiac strain, and are compatible with indefinite survival except for the possible superimposition of a subacute bacterial endarteritis, large fistulae lead to progressive heart failure, and the larger the fistula and the closer to the heart, the sooner is this likely to supervene.

Of greater clinical interest than the stab wounds and bullet wounds of axilla and groin (the commonest sources of arteriovenous fistulae), are the operative injuries causing such communications. Arteriovenous fistulae, after transfixion of vessels, have been reported in the thyroid, splenic, renal, and other vessels. In the past two decades, the great frequency with which the intervertebral discs have been explored with sharp instruments through relatively small incisions has led to a considerable number of arteriovenous fistulae, usually of the iliac vessels. The curette, passing too far anteriorly, produces an injury to the vessels; this is small enough to prevent exsanguination and, in some instances, no undue bleeding has been noted by the surgeon. At times, signs of a massive retroperitoneal hemorrhage appear at once and give the clue to the situation. In other cases, abdominal signs are totally absent and there is a slow, or more or less rapid, development of the evidences of a major arteriovenous communication. Heart failure in a patient without previous cardiac history, who has had an operation for a ruptured vertebral disc in the recent past, should lead at once to the suspicion of a traumatic or arteriovenous fistula.

For some time, the only safe and effective treatment of arteriovenous communication has been complete excision of the fistula. This can now be accomplished in all cases without the sacrifice of the major artery involved, either by suturing of the defect, or by prosthetic replacement of a segment. In most instances, the vein can be salvaged as well.

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